amber-colored fluid. It is of alkaline reaction and contains the pancreatic ferments. Usually after waiting a short time, and after repeated aspirations, a golden-yellow fluid (containing bile) appears. This has no diagnostic significance. If, however, after patient waiting and aspirating only pancreatic juice but no trace of bile appears it may be of some importance, particularly in cases of chronic jaundice. If bile is present in the duodenal contents a complete occlusion of the common bile-duct can be excluded. If the bile is entirely missing and pancreatic juice is present it points to the seat of the obstruction above the common duct."

I would like to add that absence of both bile and pancreatic juice, as already stated above, speaks for a mechanical obstruction of the common duct just above Vater's papilla, thus blocking the entrance into the duodenum of either bile or pancreatic juice (Case B.). In these instances it is advisable to ascertain positively the position of the capsule end of the tube in the duodenum by the milk test or Rœntgen rays, as there is otherwise very little to go by. In one case of probable neoplasm of the pancreas with secondary involvement of the liver, not mentioned in the table, there could be found no bile in the duodenal contents after systematic aspiration, while the duodenal bucket left in the digestive tract overnight returned filled with bile, the thread likewise showing bile for several inches near the bucket. There was no jaundice present. It is difficult to explain this occurrence. Possibly the tumor in the standing or sitting postures of the patient interfered with the outflow of bile while the obstacle did not exist in the recumbent position.

DISSEMINATED SCLEROSIS.

ITS FREQUENCY COMPARED WITH OTHER ORGANIC DISEASES; ITS ETIOLOGY AND PATHOGENESIS; THE TYPES AND DIFFERENTIAL DIAGNOSIS OF THE DISEASE; ITS COURSE AND TREATMENT. A CLINICAL STUDY OF NINETY-ONE CASES.

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Disseminated sclerosis, or insular sclerosis, is a disorder which is still rarely diagnosed in this country. Nevertheless, omitting the syphilitic and parasyphilitic processes, it is the most common organic nervous disease. Byron Bramwell made the diagnosis

100 times in the examination of 5825 cases; Bruns, 70 times in 5500 cases; Williamson, of Manchester, 61 times in 2294 cases. In three successive years there were in the National Hospital for Paralyzed and Epileptics in London 2568 cases of nervous disease, of which 159 were diagnosed disseminated sclerosis.

On the other hand in 1902 the following statistics concerning the frequency of this disease in the private and hospital practice of American physicians were revealed before the New York Neurological Society:

Dana: 3000 cases (private), disseminated sclerosis, 10 cases.

Dana: 600 cases (clinic), disseminated sclerosis, 2 cases; in Bellevue, 12,000 general cases annually, disseminated sclerosis, 2 cases yearly.

Collins: 5508 cases (clinic), disseminated sclerosis, 18 cases.

Sachs: 2000 cases, disseminated sclerosis, 15 cases.

Starr: 10,056 cases, disseminated sclerosis, 27 cases.

Fränkel: In Montefiori Home in ten years, disseminated sclerosis, 18 cases.

Such statistics and others that one of us cited in detail in a paper before the Association of American Physicians in 1906 would seem to show that the disease is three or four times more frequent in Europe than it is in this country. This seems paradoxical, inasmuch as the majority of our patients seen in dispensaries and hospitals are Europeans.

In the course of examination of approximately 12,000 individuals presenting themselves as subjects of nervous diseases in private, dispensary, and hospital practice we personally have encountered disseminated sclerosis 91 times. It becomes necessary to believe, therefore, that the process is actually more common in Europe than here, but we think that this alone does not explain the extraordinary discrepancy. We believe that here the diagnosis is not made generally unless the patient presents a majority of the symptoms which constitute the classical form of the disease, whereas in Europe the diagnosis is frequently ventured even though none of the classical symptoms or signs is evident.

Despite the careful study of disseminated sclerosis that has been made, especially in Germany and Great Britian, absolutely nothing is known of its causation. In isolated instances it seems to stand in relationship to infectious diseases, to exposure, exhaustion, trauma, and other environmental circumstances. Such factors, however, act probably only by exciting some preëxisting constitutional tendency into activity, or by favoring the activity of some unknown metabolic or infectious toxin. We throw no light upon this question; few of our cases have come to the autopsy table, so we have no pathological findings to report. Our paper deals with the clinical observation of 91 cases in which after careful deliberation the diagnosis of disseminated sclerosis was made.

Etiology. The age of onset has been recorded in 85 of our cases: 10 (12 per cent.) occurred before the sixteenth year; 65 (76 per cent.) occurred between the sixteenth and thirty-fifth years; 10 (12 per cent.) occurred between thirty-five and forty-three; 88 per cent. occurred before the thirty-fifth year, almost the exact percentage of Bramwell's 110 cases (85 per cent.). The process unquestionably makes itself evident most commonly in youth and in early adult life. We have observed but 3 cases occurring before the tenth year where the diagnosis was ventured, though in none of these was the evidence conclusive. Totski claims to have found it present before the age of six in 19 cases, and other competent observers have recorded cases occurring in early childhood. The diagnosis in these early cases can scarcely be made with certainty, however. Only their subsequent career, their course and development, or the findings at necropsy can establish the diagnosis.

Heredity as a factor was not noted in any of our cases. It is not improbable, however, that close and systematic examination would have revealed neuropathic foundations in many of them. Reynolds has reported two instances in which the familial or hereditary factors were of great significance. In the first instance three children of a family developed the disease in the thirtieth, thirty-seventh, and thirtieth years respectively. In the second instance two sisters displayed the disease, one in her twenty-fifth year and the other in her twenty-ninth. Mettler reported one case of a man, aged thirty-two years, with fully developed evidences of disseminated sclerosis, whose paternal grandparents were first cousins and congenital deaf-mutes. The patient himself was the weaker of twins. Klausner reporting 126 cases of the disease found 31 in which heredity undoubtedly played a role; 28 of our patients were native-born Americans, 13 were British, 20 were Russian Jews, 13 were of scattered nationality; 60 per cent. were unmarried; males made up 57 per cent., females 43 per cent.

Causative Factors. 1. *Occupation.* In our cases we have found such great diversity of occupation that we cannot conceive any relationship between occupation and the development of the disease. Oppenheim lays great stress upon this etiological factor. He particularly insists that metal workers and machinists are prone to develop disseminated sclerosis, and he believes that metallic poisons produce the disease. If his assumptions were correct, however, we should expect to find disseminated sclerosis much more frequently than we do among this class of workmen. We should also have to attribute almost a specific action to such metallic poisons, and we might be permitted to assume that it played a part in the development of all cases irrespective of occupation; 4 of our cases were machinists and brass-workers and 4 were jewelers working in brass, gold, and silver. On the other hand 18 were women whose activities were confined solely to the home; 7 were school-children; 13 were clerks, stenographers, actors, etc., and the remainder had various occupations. It is reasonable to believe that occupation, like exposure, fever, and environmental factors, occasionally plays the part of activator of the disease which until then had lain dormant.

2. *Trauma.* Much the same may be said of trauma. Many observers insist that accidents and injuries are responsible for the disease. In only 4 of our cases could any such relationship be established. Recently the probable role that trauma plays in the development of the disease has become very important, because of the question of liability in accidents and their consequences. Pickenbach cites a case that illustrates this:

A man, aged forty years, on leaving his work tripped down stairs and struck his right arm, tearing or straining some muscles. Examination after four months' treatment revealed limitation of active and passive shoulder movements and atrophy of the shoulder-blade muscles. No other injuries or damages were mentioned at this time. For two months massage, passive motion, and electricity were employed, after which he was considered partially capable of performing work. Three years later he made claim against his industrial insurance company because of incapacity in working on account of pain in the shoulder. The testimony of a specialist accompanied the claim and showed the subjective as well as the objective signs of a chronic spinal disease, evidently disseminated sclerosis, that left the patient totally unfitted for work. The argument in the case naturally was whether or not the accident of two years previous was responsible for the development of the spinal disease. Pickenbach believed that there was no relationship between the two: (1) because the patient had not injured his skull or his spine, and (2) because two years had elapsed before the symptoms of the nervous disease showed themselves. He was not inclined to consider the shoulder muscle atrophies as due to anything else than the shoulder injury. Perhaps the suprascapular nerve had been torn at the time. He is unable to understand how an ascending peripheral neuritis could bring about a central nervous system disorder unless there were already present other conditions that would permit of the development of decided changes in the nerve cells.

He goes on record as saying that he agrees with Kienboeck, Kurt Mendel and Schuster, Vulpius and Ewald, who believe that the development of a spinal disease after trauma is very rare, and that heretofore too much importance has been placed upon trauma as an agent in exciting the latent disease into activity or in aggravating an already existing central nervous system disorder. Vulpius and Ewald further come to the conclusion that pathologic-anatomical processes of central nervous system diseases cannot be influenced by a peripheral trauma. There is no ascending neuritis.

In some instances the trauma may have resulted because of the unsteadiness or insecurity due to an already existing disseminated sclerosis. There is no doubt, however, that trauma may introduce the disease. Frequently after the lapse of a few weeks or even a few days after the injury the suggestive signs and symptoms of disseminated sclerosis appear. Marburg suggests that there may have occurred a molecular or vascular damage in the central nervous system; that the delicate lymphatic walls have been lacerated, and that perhaps even minute hemorrhages have occurred. Nothing definite is known about this, however; attempts have been made to reproduce the condition by experimental trauma, but they have never been successful.

3. *Fatigue, Cold, and Exposure.* Exposure, wettings, and chills have been generally accepted as closely related to the development of disseminated sclerosis. Krafft-Ebing considers refrigeration one of the most significant factors in the etiology of the disease. Leyden and Goldschneider concur in this belief. Oppenheim, Cassirer, and Finkelburg believe that the nervous disease may be awakened by a chill or wetting. E. Müller attributes no close relationship between refrigeration or trauma and the disease, but he admits that it may play some role. Eichhorst states that "the actual significance of cold or wetting is not known, nevertheless there exists not a small number of cases of disseminated sclerosis in which this factor is the only one to be considered."

4. *Intoxications and Infectious Diseases.* Oppenheim is the champion of the view that metallic poisons play some part in the production of the disease. He found lead the commonest of these agents. Cases have been reported where zinc, mercury, copper, and other metals seem to be concerned in the production and development of the disorder. The intimacy of the relationship of such factors as these may be strikingly illustrated by the case reported by G. Etienne. Eight days after having been found unconscious from the inhalation of carbon monoxide the patient, a man, aged thirty years, developed paresthesias, and subsequently all the signs and symptoms of disseminated sclerosis.

The role of the infectious diseases in the production of disseminated sclerosis is well recognized. Typhoid fever, measles, scarlatina, diphtheria and pertussis have all been regarded as important etiological factors. The same is true for influenza, malaria, and rheumatism.

In none of our cases have we been able to detect an infectious disease as the causative or exciting agent. While many of our patients had experienced various infectious disorders in their infancy and childhood, in most cases years had elapsed before the first sign of the nervous disease appeared.

A well-known fact which we can corroborate is that syphilis appears to play no role whatever in the etiology of the disease. That hereditary syphilis is responsible for some cases, as Moncorvo suspects, we have never had the opportunity of observing.

5. *Physiological Causes* (Menstruation, Pregnancy, and Parturition). These events, always associated with more or less trauma, unquestionably have an important place in the etiology of disseminated sclerosis. Even puberty, with its lesser damages, has been considered responsible for the disease in a few cases.

We have not been able to fasten the responsibility for the production of the disease nor for its aggravation upon any of these factors, however. We cannot believe such events cause the disease, nor can we believe that they or the occupations, trauma, infections, and all other exogenous factors play a more important role than that of releasing a dormant or hitherto quiescent process. That some exogenous factor may be discovered eventually, proved specifically responsible for this peculiar pathological condition, is possible though problematical. At present we have not the slightest clue as to its nature.

PATHOLOGY AND PATHOGENESIS. The sclerotic areas may be few or many in number, irregularly distributed throughout the entire central nervous system, the nerve roots and nerve trunks. Even in cases where the symptoms suggest the involvement of a limited or localized area of the brain or cord smaller and milder patches are discovered disseminated throughout the cerebrospinal structures. Erb has stated his belief that a strictly cerebral or spinal type of the disease is very uncommon.

The patches appear as irregular but sharply outlined islets of a translucent grayish-red color. This color, contour, and consistency depend upon the age of the patch. In recent cases they are translucent, gray or gray-brown, soft and gelatinous, while in older cases they are glistening and firm to the touch. They vary greatly in size, from minute affairs that can scarcely be discovered without magnifications to enormous ones in which the area may be several square inches.

The distribution of the lesions has been the subject for study by many observers during the past few years, and several important facts have been brought out. For example it had always been generally considered that the pathological process confined itself, as a rule, to the central nervous system proper, the structures in which glia was a constituent. Now, however, careful study of the changes in the peripheral nervous system reveals the fact that it, too,may be attacked. Practically all the cranial nerves have been found affected; changes have been observed in the spinal roots and even in the trunks of the nerves.

Recent investigations have endorsed the views of many older observers that certain sites are more frequently attacked by the disease than others. Many years ago Charcot stated that the lesions were more numerous in the white matter; that they display a preference for the ventricle walls and for the intraspinal but not the extraspinal portions of the nerves. Erb found the centrum ovale, the walls of the lateral ventricles, in fact of the entire ventricular system, and the white columns of the cord the favorite seats of the disorder. Strümpell, Gowers, and Risien Russell have endorsed these views. The old belief of Charcot that the cerebral and cerebellar cortex were rarely attacked has been maintained to the present day. Gowers believes that the process never begins in the gray structures, though occasionally it invades these. Risien Russell calls attention to the fact that though the cortical gray is only infrequently the seat of the process the gray matter of the basal ganglia is a favorite locality for it.

These observations would seem to support the view we hold, with others, that the structures bathed by the cerebrospinal fluid are the ones attacked. Tredgold recently has stated that the patches "are most plentiful in that portion of the centrum ovale adjacent to the lateral ventricles; indeed, in many cases they are in contact with and seem to extend inward from the ventricular walls precisely as they do in the cord from the fissures." Shoyer from his interesting anatomical study of two cases of disseminated sclerosis arranges the sites of the process in the cord as follows: (1) about the posterior fissures; (2) about the anterior fissures; (3) about the central canal; (4) about the entry and exit of the roots; (5) about a point in the lateral margins of the cord. He assumes that these are produced by a poisonous agent conveyed by the cerebrospinal fluid.

The work of S. P. Kramer upon the circulation of the cerebrospinal fluid unconsciously supports this view. He has injected stain into various localities of the cerebrospinal system of the living dog, and has demonstrated "the long-known though often forgotten fact that the subarachnoid cavity does not extend over the cerebral hemispheres." When methylene blue is injected into the subdural space over the cerebral cortex it remains confined to that portion of the brain above the tentorium. When it is injected under the dura in the cerebellar region the entire surface of the cerebellum, pons, bulb, upper cord, the base of the brain, and the cranial nerve sheaths are stained. When, however, the injection is made into the subarachnoid cavity of the spine the following structures are stained: (1) the outer surface of the cord in the vicinity of the injection site; (2) the lower end of the spinal cord; (3) the structures about the central canal of the spinal cord, fourth ventricle, aqueduct, and lateral ventricles; (4) the cerebellum; and (5) the sheaths of the cranial nerves. Kramer's experiments seem to prove conclusively that the direction of the current of the spinal fluid in the subarachnoid is downward, then into the ventricular system, entering the cerebrospinal canal somewhere near the termination of the cord, then ascending the canal to the fourth ventricle, and through the aqueduct to the lateral ventricles. Such a circulating medium appears to have nothing to do with the surface of the cerebral cortex. Furthermore, the spinal nerve roots outside the dura are not stained, so there is evidently no lymph connection between the spinal subarachnoid space and the nerve sheaths. Assuming, therefore, that the poisonous agent circulates in the spinal fluid it is reasonable to believe that only these structures washed by the fluid become the sites of the diseased process.

Microscopic study of the acute patches reveals characteristic changes in the sheaths and in the axis-cylinders themselves. The diseased patch is usually sharply defined or delimited from the surrounding healthy structures. The border, however, may be less involved than the centre. E. W. Taylor has described patches in which there was no such border or intermediate zone; there was abrupt transition from the diseased to the healthy tissue.

In the border there may be observed various degrees of disturbance to the sheaths and axis-cylinders. The sheaths may be swollen, irregular, and knobby, while phagocytic cells may be observed plentifully, carrying away the products of fatty degeneration to the perivascular lymph spaces. Such spaces are often crowded with lymphocytes, leukocytes, and plasma cells, suggesting a distinct inflammatory reaction.

The axis-cylinders evidently are more resistant than the sheaths to the action of the toxic agent. Patches have frequently been described in which the sheaths had disappeared entirely, leaving the axis-cylinders intact. For a number of years this was considered the peculiar hall-mark of the pathological process, and even today, when we know that these axis-cylinders eventually are destroyed, we regard this peculiar resistance as characteristic of disseminated sclerosis.

The changes occurring in the axis-cylinders have been carefully studied and prove to be similar in character to those observed in the sheaths. They become swollen and lose their capacity for staining clearly; they become thin and spiriform; they show fusiform thickening. Definite disintegrative changes occur in certain portions of the axis-cylinder while other portions of the same fiber remain normal. The fibrils may remain intact even though the axis-cylinders are intensely swollen, but they too finally succumb.

These phenomena accompanied by vascular changes are regarded by Gombault as a "periaxial neuritis." The acute patches produced in this manner and the subsequent changes, chiefly glial and vascular in character, are defined by Marburg as "encephalomyelitis periaxialis scleroticans." His views are in the main held by G. Oppenheim, Tredgold, Lejonne and Hermitte, Maasz, Risien Russell, and others. The actual sclerotic phase occurs probably after the acute condition. With the removal of the degenerated products by the phagocytes, numerous spaces or interstices are left, giving the patch its "areolated" appearance as Redlich describes it. It is at this time that the glia proliferation attracts attention. Previous to this time no increase in fibrillar structure can be observed, though there may be proliferation of the glia cells. At times a scarcely visible webbing of young neuroglia can be discerned woven throughout the affected parts. Eventually there is a heavy glial growth and a thick fibrillar network is formed. Dense masses of interstitial tissue are found running parallel to the involved fibers in the patch. When the gray structures are affected a thick mesh composed of small glial cells and fibrils is seen branching in all directions from the denser and older centre. An ancient patch in which the glia has shrunk, and in which various changes in the vessel walls may have occurred, generally exhibits profound destruction to the axons and sheaths. It is only rarely the case that the axis-cylinders persist throughout this stage of glia proliferation, though this may actually occur.

Much discussion has arisen concerning the condition of the bloodvessels in the diseased patches. Many observers have found no significant changes, while others have described every degree of involvement of the vessel walls from simple nuclear increase and scarcely noticeable thickening to the densest thickening with hyaline degeneration and occlusion of the lumen.

What remains the most perplexing feature after all, however, is the absence of secondary degeneration in the involved tracts. Schultze has endeavored to explain this as due to the integrity of the axis-cylinders; that as long as they remained functional secondary degeneration would not occur. At present, however, we know that even with total focal destruction of the nerve fibers, sheath, and axis-cylinders there may be no secondary degeneration. Marinesco and Minea recently offered an interesting explanation concerning this phenomenon. They believe that systemic degeneration occurs, but that the degenerated fibers are absorbed and therefore are not to be observed. This idea is supported by the observations of others who have been able to note individual fibers in the columns, undoubtedly showing degenerative changes. Still others have recorded cases where the degeneration of entire tracts was pronounced.

THE CHARACTER AND ORIGIN OF THE PATHOLOGICAL PROCESS. We know no definite or adequate explanation of the nature of the process. The essential question of which structure is primarily involved has never been answered. If we possessed such information we should soon after, in all probability, discover the nature of the diseased process. The four well-known hypotheses concerning this problem are still with us and each has its supporters.

1. *The Parenchymal Theory* (Adamkiewicz, Huber, Redlich, Richien Russell, Borst, Thomas, and others). The neurons themselves are initially involved. Through the action of a toxic agent circulating in the blood or the cerebrospinal fluid there occurs focal destruction of the sheaths and axis-cylinders. Subsequently there is secondary proliferation of the glia, and eventually structural vascular changes may develop. New evidence has recently been offered (Siemerling and Raecke) which appears to support the idea that the process is a definite parenchymatous inflammation inaugurated by capillary hemorrhages.

2. *The Interstitial Theory* (Charcot, Leyden, Wernicke, Erb, Goldschneider, Gowers, and others). This is the oldest of the views, and it is summed up by Charcot's definition "a primary and multilocular chronic interstitial myelitis or encephalitis." The interstitial tissue is the initial seat of the pathological process; the changes in the parenchyma are secondary. Leyden and Goldschneider are of the opinion that the whole process is a type of chronic myelitis.

3. *The Multiple Gliosis Theory.* Strümpell regarded the disorder as a multiple gliosis, owing its existence to some congenital or inherited tendency to proliferation of this tissue. Here too the glia is the initial structure implicated, but the disorder is not inflammatory in character. Strümpell compares the disease to multiple neuroma, lipoma, and others in which congenital influences evidently play an important role. This hypothesis is the only one which assumes an endogenous cause for the disease. Such an inherited tendency may be alone sufficient, but exogenous influences may give the impetus to development of the process. E. Müller and Byron Bramwell support Strümpell's idea.

4. *The Vascular Theory* (Rindfleisch, P. Marie, Ribbert, Bastian Popoff, Buss, Hess, Williamson, and others). This theory attributes the disease to a primary disorder of the bloodvessels, with secondary changes in the parenchymal and interstitial tissues. Tredgold rejects it, saying that local areas of necrosis as a result of vascular disease have not the sharp, punched-out appearance of those in disseminated sclerosis. As a matter of fact, vascular changes are rarely prominent in this disease, while they are pronounced in definite vascular disease such as syphilitic endarteritis. In many cases of disseminated sclerosis no vascular changes whatsoever are to be found.

Notwithstanding the fact that knowledge concerning the origin of the disease is quite indefinite, we are ready to accept the view that it is essentially a parenchymatous degeneration produced by a toxic agent. Whether this be a degenerative process from the very beginning or whether it be preceded by an actual inflammation remains to be discovered.

Types of the Disease. On attempting to classify types of the disease we meet with considerable difficulty. Our classification must be either anatomical, physiological, or clinical, and none of these lends itself to definite division. That is to say, we can no longer speak of a cerebral, a cerebellar, or a spinal type unless we mean that the intense or the pronounced damage has evidently occurred in these localities, although we recognize evidences of lesser involvement elsewhere. It is exceptional that these localities are affected alone; the disease is now regarded as a general one in which the lesions are scattered throughout the cerebrospinal system. The same difficulty is encountered on attempting to group the cases from a physiological stand-point. Should we speak of a "spastic-paretic," or an "ataxic," or "hemiplegic" form of the disease we must bear in mind that only rarely do these disturbances occur, uncomplicated by symptoms of involvement elsewhere.

Nevertheless, both of these classifications, inaccurate as they must be, are not without value to us, for they reveal the widespread distribution of the process, on the one hand, and the great variety of symptoms on the other.

Depending upon the site and the intensity of the lesion, practically every symptom of involvement of the nervous system may occur. They may be so slight that their existence may remain unrecognized for months; the patient himself may be unaware of them or he may disregard them. Minor disturbances of muscular power, a tendency to fatigue too readily, a slight incoördination or loss of skill, may not excite his attention until they tend to incapacitate him. This is true, likewise, of the sensory disturbances, paresthesias, blurred and double vision, headache and dizziness, and many others, particularly when they are of temporary and short duration.

On the other hand the disorder may be introduced without the slightest warning by a complete and sudden apoplectiform hemiplegia, which may be accompanied by aphasia.

We have found in the large majority of our cases that the earliest manifestation was weariness and weakness in the legs. The patients describe this "leg weariness" as a feeling of heaviness in the feet and an abnormally early tiring and fatigue. Frequently there is associated a weakness of the bladder; there is inability to hold the urine as well as usual, and there may be urinary incontinence at times. Invariably when the patients presented themselves there was already established an involvement of the motor mechanism of the legs and bladder, which varied from a mild spasticity to a totally incapacitating spastic paralysis. It is quite extraordinary that we report eighty-three of our cases of this type, not one in which the arms were involved in a similar manner. And equally remarkable is the fact that we have never encountered a case in which the paralysis was of the lower neuron type.

The variability in the manner of development is as great as that of its intensity and impairment. For a long time, perhaps years, there may be no subjective manifestations. Not infrequently the patient tells of occasional cramps in the legs or a "drawing up" of the legs at night. There may be but slightly disordered gait, due to a characteristic stiffness which, in severer form, may be associated with weakness and unsteadiness. In other cases the gait may be profoundly impaired and in a short time locomotion may become impossible. Under such circumstances the rigidity may be extreme and the patient becomes unable to lift the feet from the floor because of flexor spasm. Eventually, unable to stand, he becomes the bed-ridden victim of flexor and adductor contractures.

The bladder and rectal sphincters seldom escape in disseminated sclerosis of this type. Here again all degrees of impairment are met with, although, as a rule, they are slight in the beginning and are due to hypertonicity. The earliest difficulty is the inability to retain the urine for any considerable time; it becomes imperative to release the bladder upon its earliest demands. Retention often occurs and the spastic contraction of the sphincter may be sufficient to prevent the entrance of a catheter into the bladder. In later stages and in fully developed cases of the lower spinal type of the disease incontinence is present practically always. Involvement of the rectal musculature occurs much less frequently.

Disorders of the cutaneous sensory mechanism were present in less than 10 per cent. of our cases. Most of these were subjective feelings of numbness and tingling in the extremities. Pain as a symptom was rarely encountered. It is an interesting fact, in accord with most writers, that the shooting pains and other crises so common in tabes practically never occur. Objective sensory disturbances were discovered very rarely, and even in those cases where paresthesias were pronounced it was the exception to find noticeable defects in the acuteness of the cutaneous sensibility. Cases occur, however, where there is marked sensory disorder in the hands and feet. Deeper sensibility was impaired in very few of the cases seen by us. Astereognosis was recorded in four of them.

We wish, therefore, to classify a group of cases of disseminated sclerosis which was by far the commonest in our series as the spastic type; characterized by hypertonic paresis or paralysis of the legs, usually accompanied by bladder weakness, and, infrequently, by disturbances of the cutaneous and deep sensibility. Quite often no other signs were present, but usually there were evidences of involvement of other parts of the nervous system. Nevertheless the characteristic impairment was the involvement of the legs and bladder. This permits of a convenient clinical grouping; 65 per cent. of our cases come under this division.

A second type of the disease may be considered as the ataxic.

Here the dominating feature is the incoördination or unsteadiness due to ataxia and tremor. Arbitrarily we may place in this group those cases in which the chief disorder is ataxia, vertigo, intention tremor, scanning speech, and nystagmus. These symptoms rarely occur singly. Usually they are all present in the same case, and quite often they are accompanied by manifestations of more or less severity due to involvement of the spinal cord. This is the cerebellar form of disseminated sclerosis; 25 per cent. of our cases were of this group, which approaches the classical type of the disease described by Charcot.

Ataxia is not infrequently the initial symptom of bulbar and cervical involvement. Such cases may simulate tabes dorsalis; the disordered gait and station may be identical with that of tabes because of involvement of the kinesthetic pathway. More commonly, however, the ataxia is of the cerebellar type, uninfluenced by vision; that is to say, it is not made worse by closure of the eyes.

Intention tremor is more frequently encountered. The nature of this characteristic symptom has given rise to considerable speculation, particularly concerning its relation to ataxia. At times the tremor becomes so pronounced that it results in marked incoordination. Strümpell considers it a genuine ataxia and Risien Russell believes it is due to "a failure of synergic action of muscles in purposive movements, consequent on an unequal degree of innervation both of the muscles directly concerned and of their antagonists." The tremor usually accompanies movement and is seen, therefore, more commonly in the eyes (nystagmus), head, and hands. It is well known, of course, that it becomes most pronounced on effort or fixation and in muscles that have been previously fatigued. The innervation of the trunk muscles is quite frequently affected and the station and gait may be profoundly disordered by this asynergia alone.

A great variety of speech defects have been described. These range from the slightly monotonous speech (bradyphasia) to disturbances which are truly ataxic. The characteristic disorder, however, is the slow, scanning, syllabic speech due to abnormal intervals between the words and even between the syllables. The cause of this is an unusually rapid fatigue of the speech and respiratory muscles and an inability to adjust perfectly the relation between the action of the expiratory muscles and those of the larynx. This rapidly developing weariness and fatigue is well illustrated in one of our cases:

Patient is M. B., a young girl, unable to speak any considerable sequence of words because of loss of power. She is also unable to hold her breath after a full inspiration for more than a few seconds. In attempting to sing any moderately high tone she can sound it, but it exhausts almost at once. These disorders, in addition to a slight oscillatory nystagmus, were the only symptoms present; nevertheless, we feel justified in regarding them as evidence of a beginning disseminated sclerosis.

Very rarely the speech becomes almost entirely disorganized. It may be of a paralytic character, and when accompanied by disturbances in coördination of the swallowing, breathing, and heart mechanism may be considered due to a lesion in the bulb.

Nystagmus may be considered as fundamentally a fatigue phenomenon, due to an incapacity for even slightly sustained effort, because of abnormally quick fatigue (Uhthoff, Marie, Strümpell). Essentially also it is an intention tremor (Charcot), similar to those we have been describing. We shall discuss nystagmus more in detail in a subsequent part of this paper.

The third group is characterized by hemiplegia of evident cerebral origin, identical with that occurring as the result of the occlusion of a cerebral artery. This hemiplegic type occurs relatively infrequently. Developing gradually, as a rule, but at other times with apoplectic suddenness a paralysis of one side of the body occurs, varying in intensity from a slight weakness and loss of skill in performing fine movements to complete incapacity. Such a disorder due to disseminated sclerosis is usually described as of fleeting character. After a few weeks or even after a few days the function may be restored in large part or completely. Our cases (7 per cent. of the series) were not so fortunate. Months and years elapsed yet no improvement took place.

Again it becomes necessary to remark that the hemiplegia may exist alone. No other characteristic evidence of disseminated sclerosis need necessarily be present, especially if the disease is being introduced in this fashion. The paralysis may develop overnight in an individual otherwise apparently normal. The subsequent career of the case is necessary, as a rule, before a definite diagnosis may be permitted. More usually the hemiplegia arises in cases that had been recognized previously as disseminated sclerosis, or which upon subsequent examination show significant signs of involvement of other parts of the nervous system. An instructive illustration of the hemiplegic form of the disease is the following case:

A strong, well-developed girl, aged nineteen years, a champion swimmer, noticed a progressing awkwardness in her hands, which had been preceded for two months by a painful coldness in the hands. She dropped articles from her hands when her visual attention was not directed upon holding them.

Her previous history was negative except for two accidents from which she apparently fully recovered. When sixteen years old she struck her head while diving and immediately felt a "pins-and-needles" sensation down her spine, but no apparent damage was done. During her seventeenth year she was knocked senseless in a basket-ball game and felt too ill to attend school the following day, but was soon as well as ever.

Soon after the disability in her hands, weakness of the left leg developed so that it dragged in walking. She suffered also from an acute coldness in all the extremities. At the same time it was observed that she did not fully appreciate pin-pricks in the hands and arms. Three months later she became irritable, emotional, and unable to walk or stand. After seven weeks in bed she improved so much that she was able to take long walks; the left arm and hand, however, had become increasingly disabled, so that they were now stiff. After being urged she went into the ocean to swim, the idea being to increase the function of the rigid arm and hand. The result was disastrous, as there developed a rigidity of the left leg and loss of bladder control, as well as ataxia in the right arm. In short, within a period of six months a vigorous athletic girl had developed a hemiplegia which has persisted to the present time, a period of three years. Her mentality is good but she is emotionally unstable. In speaking, words flow faster and faster, so that at the end of a statement there is an explosion of sound accompanied by exaggerated movements of the face and irregular movements of the right arm. The spinal fluid is normal.

As a matter of convenience we shall now consider the signs and symptoms of involvement of other parts of the cerebrum and the cranial nerves. This includes psychic disorders as well as disturbances of the optic and auditory pathways. Disseminated sclerosis may exist without any of these, particularly when the pathological process is confined largely to the spine and bulb. As a rule they are present, more or less prominently, in all cases where the cerebrum and cerebellum chiefly are affected, and this is particularly true of involvement of the optic tract.

Ocular Phenomena. The recognition of the changes occurring in the visual apparatus is of the utmost importance in the matter of diagnosis. It not infrequently happens that they may be the only manifestations of the disease for months and even years before other characteristic signs appear elsewhere. Windmüller, for example, records a case in which acute retrobulbar neuritis occurred seven years before any other symptoms could be distinguished. Moreover the ocular changes are among the commonest of the diseases, as Uhthoff has disclosed, and they may be considered almost characteristic or at least strongly corroborative of disseminated sclerosis alone. The most typical are the changes that occur in the acuity of vision, the disorders of the visual fields, and the alterations in the appearance of the disks.

Visual Acuity. A slight blurring of vision with occasional flashes of light before the eyes may be the earliest manifestation of the disease. As a rule this is only a transient process which may not reappear for weeks or months. It usually remains confined to one eye, and it may or it may not be accompanied by changes in the disks. This tendency to improvement and relapse has long been regarded as peculiarly characteristic of disseminated sclerosis. There may be defective vision for months, limited, perhaps, to one eye, followed by a period lasting for months, during which there is apparently normal vision, then eventually the return of the disorder to the same eye, the other eye, or both of them.

The onset may be gradual and scarcely appreciated by the patient. More frequently it announces itself suddenly in almost complete blindness. Even in such cases remissions occur lasting variable lengths of time. This peculiar quality of the symptoms need not necessarily be present in every case, however; 21 per cent. of our cases had ocular symptoms, but in not one of them could a history of remission be obtained.

Visual Fields. The essential cause of the amblyopia and the disorders of the visual fields is considered to be a sclerotic patch in the optic nerve, commissure, or tract. This is contrary to the older view of Gowers, who regarded the optic atrophy as the result of a primary degeneration of the nerve elements similar to that which occurs in tabes. The more recent evidence supports the idea that the pathological process is a retrobulbar neuritis which results in more or less destruction of the nerve elements. We should expect early manifestations of involvement of the macular fibers, therefore, and this actually occurs. Central scotoma is a prominent and common disturbance in disseminated sclerosis. Uhthoff observed it in 15 of his 20 cases; Klingman, recently, in 11 of 14 cases.

The occurrence of retrobulbar neuritis in any case, but especially in the young, should at once excite suspicions of disseminated sclerosis. It is an acute process, as a rule, giving rise to damaged vision and central scotoma which show a tendency to improvement. Nicotin, alcohol, and syphilis have commonly been blamed heretofore, but in many cases it has been impossible to establish any relationship between the disorder and these agents. Such cases of so-called "idiopathic retrobulbar neuritis" have recently been studied by Fleischer, who regards them as early manifestations of disseminated sclerosis. Schultze, furthermore, examined cases in which tobacco, alcohol, and syphilis had been considered the causes and found unmistakable evidences of disseminated sclerosis in many of them.

Peripheral contraction of the visual fields, while not so common as central scotoma, occurs quite frequently. Uhthoff observed it in 11 of his 24 cases; Klingman in 11 of 14 cases.

Changes in the Disks. The characteristic change noted in the disks when the optic nerve has been implicated is atrophy. Any stage of the process may be encountered from a slight pallor of part of the disk to complete atrophy in both eyes. The entire disk may not be equally involved. Various portions appear degenerated before others; even in the earliest stages it is well known that the temporal half is earlier affected than the nasal half. There may be a great difference in the extent of involvement of the two disks; a beginning pallor alone may exist in one eye while extensive atrophy is present in the other.

The most remarkable fact, however, concerning the changes in the disks is the discrepancy which exists between the apparent degree of atrophy and the actual disturbance of vision. Pronounced pallor of the disk may be present for months and years with practically no diminution of visual acuity. On the other hand, marked involvement of the fields and visual disorders may exist when the disks appear quite normal. This peculiar lack of parallelism between the structural damage and the functional incapacity is unique with disseminated sclerosis among all the organic nervous diseases. Not only is this true on the part of the ocular apparatus, but the entire brain and cord may be the seat of extensive tissue changes disclosed by autopsy in cases whose clinical career had been extraordinarily mild.

Affections of Ocular Motility. Nystagmus when present is one of the most characteristic symptoms of disseminated sclerosis. It is one of the original triad of symptoms of the classical types of the disease described by Charcot, and even now when we no longer demand its presence to distinguish the nature of the disorder we recognize it in nearly 60 per cent. of all cases.

As a rule true nystagmus appears late in the course of the disease, though "nystagmiform" jerkings and twitchings may have existed before. Uhthoff, Baranyi, and Bartels would have us distinguish between the two. True nystagmus, which is oscillatory, swinging to and fro about a central point with rapid equal movements, is in all probability a fatigue phenomenon (Uhthoff, Marie, and Strümpell). It occurs independently of voluntary movement of the eyes, in changing the direction of gaze; the oscillations continue even in the resting stage. It becomes more intense, however, when the eyes are turned and attempts at fixation are made, particularly in extreme directions. At such time the "nystagmoid" movements appear. They are characterized by sharp, quick jerks in the direction of the object of fixation, with slower movements of return. These movements occur about three or four times a second and appear to be in every respect "intention tremors" (Charcot).

Uhthoff found external ophthalmoplegia well established in 17 per cent. of his cases. The sixth nerve was more frequently involved than the third or fourth. The disorder is not usually so severe, however; transient weaknesses of individual muscles are more likely to be met with, and these are scarcely ever maximal. Windmüller records ocular palsies, counting diplopia as an evidence of such in 70 per cent. of his cases.

Pupillary changes are uncommon. The reflexes are generally active, and only at times may a slight disparagement be observed between the activities of the two sides.

Auditory and Vestibular Systems. The cochlearis division of the auditory nerve is only rarely involved. The symptoms may vary from a slight acoustic hyperesthesia (E. Müller) to total deafness. O. Beck describes two cases in which he was able to diagnose disseminated sclerosis without any neurological data except the peculiar variations of the acoustic and labyrinthine functions. Excitability of the vestibular apparatus and capacity for acute hearing, determined by many tests, alternated with absence of excitability and complete deafness without revealing in the interval any of the typical signs of labyrinthine impairment. This variance between the subjective symptoms and the objective signs is similar to the condition frequently observed on the part of the visual apparatus; the coexistence of quite normal vision with marked pallor of the disk, and *vice versa.*

Unusual rapid fatigue of the acoustic mechanism determined by tuning-forks was discovered by Claude and Egger, and daily changes in acoustic keenness have been observed by Marburg.

Involvement of the vestibularis division of the auditory nerve is not uncommon. Vertigo is frequently an initial symptom of the disease. Nystagmus and its significance has been discussed previously.

Psychic Phenomena. Psychic disturbances of every variety have been observed from slight blunting of mental activity to profound dementia. They may precede the development of the more usual symptoms of disseminated sclerosis by months or years, their true nature, therefore, remaining unrecognized. It is very difficult to say how frequently mental symptoms occur as early evidence of the disease, for they may be of such slight significance as to escape notice, or so severe as to necessitate the confinement of the patient in psychiatric hospitals where it is not unlikely their true nature could only have been determined by the subsequent appearance of other characteristic evidence.

Changes in the character and disposition may occur; an unusual cheerfulness, an uncustomary tendency to laughter entirely out of proportion to the exciting cause. A distinct euphoria is common, accompanied by more or less boisterous laughter. Ten of our cases presenting mental symptoms behaved in this fashion, and they revealed, in addition, impairment of memory, judgment, observation, and attention. As a rule there was always to be observed a heightened sensitiveness or increased excitability, though in none of them did this ever become a delirium or mania.

In three cases pronounced apathy and depression were early symptoms.

COURSE AND DEVELOPMENT. Disseminated sclerosis reveals itself gradually, as a rule, developing so insidiously that it frequently escapes observation for months. We must not lose sight of the fact, however, that it may be inaugurated abruptly, with apoplectic manifestations, although, as we have pointed out, this manner of onset is very unusual.

Once established the disease runs a chronic progressive course, during which there may be remissions and even apparently complete recovery. Most observers lay stress upon this. It has become an accepted fact that remissions are characteristic of the disease. We have no intention of disputing this; nevertheless, in our cases it has not been observed except in a few instances when slight improvement occurred. With these exceptions they were all of the chronic, progressive type of long duration. The duration of the disease is very indefinite. It is apparent, of course, that years may elapse before death, which is usually due to complications or intercurrent diseases.

Diagnosis. The recognition of the fully established Charcot case presents no very great difficulties as a rule. With all the cardinal features, or even a few of them, there is little possibility of error. Tremor, nystagmus, and incoördination defects, especially in the young, may be considered due to disseminated sclerosis, keeping in mind, however, the important reservation of cerebellar tumor or cyst. It is vitally important that we do not overlook these because of the relief now possible by early surgical intervention.

Cerebellar Tumor and Cyst. The difficulty in the differential diagnosis is dependent upon the fact that a small cerebellar cyst or a slowly expanding tumor before being able to raise the intracranial tension behaves exactly as a sclerotic patch. The difficulties may be illustrated by this case:

J. H., aged nineteen years. In June, 1912, his gait became unsteady; people believed him to be intoxicated. Two months later he began to vomit after his meals and soon began having headaches all day long. Two months later the headache and vomiting diminished in intensity, but he then became disturbed by vertigo. On examination he presented static and dynamic ataxia, tremor, slurred speech, and nystagmus. The disks seen in the early phases of the process showed clear and there was no fault in the vision. In the course of the following year the signs of increased intracranial tension revealed themselves. A definite choked disk appeared and an operation was decided upon. Cystic tumor of the right cerebellar lobe was found. The patient recovered promptly and at the time of this record has been relieved of most of his disorders.

Only one fact is of value in influencing our diagnosis and determining our course in this case: that is the presence of symptoms that must always be regarded as significant of increased intracranial tension. Headache, vomiting, and giddiness, with optic neuritis, are not at all uncommon in disseminated sclerosis; nevertheless these must remain the exceptions, and in case of doubt, as we encountered in the case above, with such symptoms present, we dare not disregard the idea of tumor until we have disproved its existence.

The absence of spasticity is of no value one way or the other. It is true that in disseminated sclerosis we encounter spastic phenomena commonly because of involvement of spinal tracts by other sclerotic patches, and in cerebellar tumor this does not occur until by expansion pressure is exerted upon the pyramidal system.

Because of the symptoms of increased intracranial pressure we diagnosed tumor which proved to be correct.

Cerebrospinal Syphilis. Of first importance in the differential diagnosis of the less well-established and aberrant forms, particularly of the spinal or spastic-paretic type, is syphilitic pachymeningitis. The occurrence of spastic phenomena in the legs, with or without sensory and bladder disorders, is due, in the vast majority of cases, to syphilis; we have come to look upon these symptoms as more or less distinctive of this disease, and especially when a history of the infection is acknowledged. The difficulty in the differentiation is not so great in advanced cases; there is usually some corroborative evidence present, as involvement of the pupils, the cranial nerves, or, perhaps, apoplectiform attacks. Without these, especially in the early phase of the disease and without additional proof of disseminated sclerosis, we have no means of deciding unless we resort to examination of the cerebrospinal fluid.

We are in possession of valuable facts concerning the spinal fluid in nervous diseases through the important studies and contributions of Nonne, Plaut, Noguchi, Kaplan, and many others. The information obtained from serological and chemical examination of the fluid is invaluable in practically every disease of the nervous system we are called upon to treat; spinal puncture is a routine necessity in all neurological practice even when we feel secure with our clinical diagnoses.

Two laws seem already to have been established: (1) a positive Wassermann reaction in the spinal fluid, irrespective of the reaction in the blood, is absolutely certain proof of cerebrospinal syphilis; (2) in disseminated sclerosis the fluid is normal in every particular, that is to say, the Wassermann is negative, and there is no increase in the globulin or the lymphocytes.

The value of this information is at once apparent. If we recall that invariably with a cerebrospinal showing a positive Wassermann reaction we find the globulin and the cells increased, at times only slightly and at others enormously, we realize that we have a means of distinguishing between the luetic and the non-luetic spinal diseases.

Kaplan in the analysis of more than 3000 spinal fluids obtained from patients having nervous diseases found 16 in which the diagnosis of disseminated sclerosis had been made with certainty, all of which possessed fluids normal in every particular. In cases where the differentiation was doubtful the laboratory analyses were considered sufficiently strong evidence to decide the question. When the fluid contained fifty or more cells to the cubic millimeter or an excess of globulin or a positive Wassermann reaction the diagnosis of disseminated sclerosis was excluded.

Whereas it is very common to find with a negative Wassermann in the fluid an increase of lymphocytes with or without an increase of globulin the reverse practically never occurs; that is to say, a positive Wassermann does not exist where there is neither lymphocytosis nor globulin increase. In but 6 of his 3000 analyses did he meet with this phenomenon, and all of these were cases of arterial syphilis.

Transverse Myelitis. In transverse lesions of the cord produced by localized myelitis, tumor, gumma or caries, or syringomyelia the differential diagnosis may require very careful deliberation. Myelitis develops quickly, as a rule, accompanied by fever and toxemia. Its sequelæ, spastic weakness, or paralysis of the legs and visceral and sensory disorders may be very severe and permanent, but is not progressive. Atrophy and fibrillary twitchings, frequent in transverse myelitis, are seldom encountered in disseminated sclerosis. Usually there are no cerebral symptoms in transverse myelitis.

Disorders of vision may be present, though they are unusual, and as recently pointed out by Ward Holden, lateral hemianopia for one eye alone is a symptom of considerable diagnostic value in cases in which visual disturbances precede the spinal symptoms.

Oppenheim and Cassirer have recently reported cases in which there developed quickly an acute ataxia with weakness of the upper extremities, atrophy of the intrinsic hand muscles, paresthesias, and objective sensory disturbances. They regarded the cases as acute cervical myelitis until subsequent developments proved them to be disseminated sclerosis.

Spinal Tumor. Disseminated sclerosis not infrequently gives rise to a series of symptoms suggesting spinal cord tumor. Nonne recently called attention to the difficulty which may arise in differentiating between them. Laminectomy performed in four cases of suspected tumor proved negative. Subsequently in two of these section showed disseminated sclerosis and disseminated confluent myelitis; and further progress of the other two substantiated the diagnosis of disseminated sclerosis. One of these was a youth, aged nineteen years, with symptoms of a transverse lesion of the dorsal spine. There were spastic paralysis of the legs and sensory disturbances to the ribs, with fever and slight delirium in the beginning of the disease. A laminectomy was performed, but no lesion was discovered. In a few months decided improvement took place, and for five years the patient has been well. The history of this case is astonishingly like that of another which we had the privilege of observing on the service of Dr. Pearce Bailey. In a few months after laminectomy for suspected cord tumor had been done the patient, a splendidly developed athlete, with a profound paraplegia and bladder and sensory impairment, recovered completely; no trace of the disorder remained except an exaggeration of the knee and ankle-jerks.

Syringomyelia. Under certain circumstances, syringomyelia may resemble disseminated sclerosis, particularly as the rather characteristic dissociation of the cutaneous sensibility may occur in the latter. It has been stated also (Russell) that nystagmus may occur in syringomyelia.

In the early months of the following case the question of the existence of syringomyelia was given respectful consideration. Only with the unfolding of characteristic evidences of disseminated sclerosis was the correct diagnosis possible.

W. N., aged twenty-seven years, a draughtsman. His history is negligible. His habits excellent. He seeks relief for weakness in the right arm, numbness of the entire left side, and some slight difficulty in starting the urinary flow. These symptoms developed rather suddenly two weeks before his admission here.

Status July, 1910. Weakness of the right arm and to a lesser extent the right leg. All tendon jerks on the right are increased; ankle and wrist clonus present. Babinski reflex in right foot. All cutaneous reflexes absent on the right. Almost complete loss of cutaneous sensibility in the left half of the body from the clavicle to the toes. Wassermann negative in the blood-serum and in the spinal fluid. Patient made almost complete recovery in ten days.

Status August 22, 1910. New attack with sudden onset involving the right arm, the right leg, and the left leg. These extremities are now spastic while on both sides of the body there is complete loss of sensibility. Improvement began almost immediately. In one month the left leg was practically well; the right side improved more slowly.

Status October, 1910. Patient, who had been in good condition, suddenly became quadriplegic and lost all sensation to the level of the shoulders. Improvement began again, more slowly than before. In January, 1911, the left arm and hand were almost normal. He was also able to use the right hand quite well. Both legs still profoundly involved by a high-grade spasticity. Right and left-sided Babinski sign is present.

Status May 5, 1911. Halting, scanning speech is present now. There is also fine nystagmus apparent. Patient is depressed and irritable.

Paresis (general paralysis of the insane). There should be no longer any difficulty in differentiating between disseminated sclerosis and paresis, which it occasionally resembles. The stumbling and trembling speech of paresis with its omitted syllables and words differs fundamentally from that of disseminated sclerosis, while the mental changes of the latter are usually not so severe as of the former. In fact the objective evidences of paresis, including at the present time the characteristic cytological and chemical findings of the cerebrospinal fluid, are conclusive proofs of the disease.

Hysteria. We believe we have not encountered the difficulty, said to exist, in distinguishing between disseminated sclerosis and hysteria. The majority of authors, however, lay considerable stress upon the similarity between them. They are practically of one voice, furthermore, in saying that the difficulty generally results in mistaking an existing disseminated sclerosis for hysteria.

The two disorders are common to youth and young adult life, and both frequently develop after a physical or psychical trauma. A widespread variability of disorders is common to both of them, involving somatic, visceral, and mental functions.

The regular, concentric narrowing of the visual fields, especially with parallel contraction of the color fields, speaks for hysteria. Optic pallor and optic atrophy of hysterical nature is impossible. True nystagmus should always be considered evidence in favor of the organic disease. Sensory changes are common to both disorders, and are, of themselves, of not much importance in the differentiation. Sharply defined anesthesias and analgesias are not usually found in disseminated sclerosis and an anesthetic cornea practically never occurs. Bladder disorders are usually indicative of disseminated sclerosis, and actual exaggeration of the reflexes, especially when accompanied by a Babinski sign, must always be regarded as proof of organic nervous disease.

A sign of great importance in the diagnosis of disseminated sclerosis is the absence of the abdominal and epigastric reflexes. This was first pointed out by Strümpell, who observed it in 67 per cent. of his undoubted cases. His views have been corroborated by many observers. E. Müller and Cassirer, for example, regard the bilateral absence of these reflexes as of distinct and useful diagnostic significance. Marburg noted its absence in 38 of his 40 cases, and interprets it as an involvement of the lower third of the dorsal cord.

Treatment. No advance has been made in the treatment of the disease if we except radiotherapy as used by Prof. Marinesco, of Bucharest. He ventured to try this therapy because of the good effects he had observed in the use of the Rœntgen rays in disseminated sclerosis and syringomyelia. He described unquestionable improvement in the tremors, the weakness, and the fatiguability. Rays of 1 milliampère intensity were applied for seven to ten minutes to the lumbar spine. No skin irritation or other complication ensued. We scarcely dare place any great hope in the agent, however, when we recall the nature of the pathological process. We must remember, too, that the improvements we may attribute to the action of the remedy may occur spontaneously.

In the meantime it is well to bear in mind the good advice of Oppenheim and Müller, that rest is essential; all fatigue is to be avoided, and only moderate exercise to be permitted. Gentle massage and sedative baths may be appreciated.

RESUMÉ. Disseminated sclerosis is encountered much less frequently in this country than abroad. This is due, in part only, to the fact that early and irregular cases frequently escape recognition here.

The fully developed, classical Charcot type of the disease is rarely seen.

Trauma, including occupation, injury, intoxications, biological crises, mental shocks, etc., play only a contributing role in the development of the disorder.

The disease is regarded by us to be a parenchymatous degeneration due to the action of a toxin circulating in the cerebrospinal fluid.

There can be no rigid grouping of "types" of the disease. Nevertheless three general groups can be assumed: a spastic-paretic, an ataxic, and a hemiplegic group.

The best single aid in the differential diagnosis is the condition of the cerebrospinal fluid. The Wassermann reaction is practically always negative. The number of lymphocytes is less than fifteen, and there is no excess of globulin. This is of special value in ruling out cerebrospinal syphilis.

The remissions which sometimes occur are singularly characteristic of this disease. In our series, however, they were rarely encountered.

The absence of the abdominal reflexes, upper and lower, is an important diagnostic sign.

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